Increased Oxidative Stress Induces Apoptosis in Human Cystic Fibrosis Cells

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Résumé en anglais	Oxidative stress results in deleterious cell function in pathologies associated with inflammation. Here, we investigated the generation of superoxide anion as well as the anti-oxidant defense systems related to the isoforms of superoxide dismutases (SOD) in cystic fibrosis (CF) cells. Pro-apoptotic agents induced apoptosis in CF but not in control cells that was reduced by treatment with SOD mimetic. These effects were associated with increased superoxide anion production, sensitive to the inhibition of IκB-α phosphorylation, in pancreatic but not tracheal CF cells, and reduced upon inhibition of either mitochondrial complex I or NADPH oxidase. CF cells exhibited reduced expression, but not activity, of both Mn-SOD and Cu/Zn-SOD when compared to control cells. Although, expression of EC-SOD was similar in normal and CF cells, its activity was reduced in CF cells. We provide evidence that high levels of oxidative stress are associated with increased apoptosis in CFTR-mutated cells, the sources being different depending on the cell type. These observations underscore a reduced anti-oxidant defense mechanism, at least in part, via diminished EC-SOD activity and regulation of Cu/Zn-SOD and Mn-SOD expressions. These data point to new therapeutic possibilities in targeting anti-oxidant pathways to reduce oxidative stress and apoptosis in CF cells.

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